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The Apexcardiogram in Ischemic Heart Disease

HOWARD H. WAYNE, M.D., *San Diego*

■ *The apexcardiogram (ACG), when recorded serially in patients with acute myocardial infarction (AMI), preinfarction angina (PIA) and stable ischemic heart disease (IHD), appeared to reflect the abnormal patterns of contraction of the left ventricle in these conditions. Thus, paradoxical bulging (dyskinesis) of the systolic wave or increased "a" wave amplitude with gradual recovery over several weeks was found in all 60 patients with documented AMI and in 18 of 20 patients with PIA. Electrocardiogram changes were noted, however, in only eight of the PIA patients. Changes in the ACG frequently antedated ischemia in the ECG. Paradoxical bulging of the systolic wave of the ACG was additionally noted in patients during the pain of angina pectoris but this promptly disappeared after the administration of nitroglycerine. Patients with classic angina often had normal resting ECG's but abnormal resting ACG's.*

In contrast to the relatively transient abnormalities noted above, the ACG remained unchanged in most patients with stable IHD during follow-up of three months to two years. Patients undergoing coronary bypass operations, however, showed immediate improvement in the ACG in the postoperative period.

These results suggest the ACG reflects the contractile pattern of the left ventricle, and may be an indirectly recorded ventriculogram. Its enhanced sensitivity and the earlier development of changes in comparison to the ECG make this a valuable tool in the study of patients with heart disease.

IT IS A COMMON clinical observation that ischemic heart disease (IHD) may exist in advanced form without clinical manifestations; indeed even in the presence of symptoms a definitive diagnosis is often difficult. Although most physicians, when confronted with classic angina pectoris, feel reasonably secure in their diagnosis even without additional clinical support, they are less certain when pain or other symptoms are atypical. Pity, therefore, the asymptomatic patient with IHD, examined routinely with just a stethoscope and resting electrocardiogram (ECG) and pronounced "well" so far as his heart is concerned when no abnormalities are detected.

Unfortunately the insensitivity of the usual tools—that is, the stethoscope, the resting ECG, and even the exercise ECG—is not generally ap-

From the San Diego County Heart Center at the Donald N. Sharp Memorial Community Hospital.

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Reprint requests to: H. H. Wayne, M.D., 2290 Sixth Avenue, Suite 203, San Diego, Ca. 92101.

preciated. This is perhaps why half of all deaths from acute myocardial infarction (AMI) are reported to occur in persons with no previous clinical heart disease[1] and why 25 percent of all AMI's are "silent" or atypical.[2,3] Coronary cinearteriography is, of course, the definitive test. Even so, discrepancies often exist between the clinical features and the cinearteriograms.[4,5] Additionally, coronary cinearteriography is neither routine nor practical to perform serially, nor is it universally available.

These problems are possibly prime reasons why many patients who are to have an AMI are not identified, in spite of warning symptoms for one to two months,[6,7] and perhaps why 60 to 70 percent of all deaths occur before they reach a hospital.[8-11] Conceivably, if earlier detection of IHD or an approaching infarction were possible, corrective measures or prophylactic medication could be instituted.

There is promise that such early identification may be possible through study of the apex impulse. Harrison, Dimond and others[12-18] have long emphasized the usefulness of precordial palpation, and numerous precordial graphic recording techniques for studying the apex movement have been described.[19-25] The best known of these techniques, the use of the apexcardiogram (ACG), is relatively simple to do. Its ability to reflect both systolic and diastolic events in the cardiac cycle as well as hemodynamic changes has been extensively documented.[26-38] Close correlation between abnormalities of the systolic contraction wave (SCW) of the ACG and abnormal left ventricular wall motion—that is, dyskinesis, akinesis and asynchrony—have recently been confirmed directly with ventriculograms and cinefluoroscopy.[39-43] Since it is recordable at the bedside or in the office, is non-invasive, and can be obtained serially in the same patient, its use in the study of patients with IHD appears promising.

It is the purpose of this paper to review some of the graphic changes we have seen in the ACG in normal persons and in patients with IHD from our study of approximately 3000 ACG's in 600 patients during the past five years. Preliminary studies have been described previously[44] and more detailed information is to be reported elsewhere.[45] Emphasis in this paper, therefore, will be on simple description of the wave form changes in various phases of IHD.

Methods

There were 215 normal persons with no history, physical findings, ECG or phonocardiographic evidence of heart disease. Ages ranged from 20 to 60 years. There were 325 patients with documented IHD based upon a classic history of angina pectoris, or an abnormal electrocardiogram (ECG) reflecting either an old myocardial infarction or significant ST segment depression. Ages ranged from 35 to 80 years. One hundred and thirty of these patients were followed serially for three months to two years. An additional 60 patients with documented acute myocardial infarctions were also recorded and followed serially.

Method of recording the ACG. This has been described in detail and is being reported elsewhere,[46] and therefore will be outlined only briefly here. The patient was placed in the lateral decubitus position; the exact center of the apex impulse was marked, and the apex pick-up was strapped precisely over this area. We have found the end-piece of a B-D Fleischer stethoscope with the diaphragm removed to be the most convenient pick-up to use. It is flat, is easily positioned and does not create artifacts as other pick-ups do.[46] Using adaptors and approximately ten inches of rubber tubing, the pick-up was connected to a pulse wave transducer (a Hewlett Packard 374 or 21051D and a Siemans calibrated transducer were used in this study). This in turn was connected to a suitable recorder. Although most of the tracings in this study were obtained on a four or seven channel Elema Schonander Mingograf, at times recordings were made on a single channel Sanborn series 500 ECG or the Sanborn Twinbeam Phonocardiograph. In general, a simultaneous ECG, carotid pulse tracing (CPT) and phonocardiogram (PCG) were obtained along with the ACG.

Since positioning of the ACG pick-up is critical, displacement after its release because of movement of the underlying skin from pull created by the rubber strap had to be carefully avoided, lest artifactual recordings could result. Oscilloscopic monitoring before the recording considerably facilitated proper placement. In addition, once positioning was achieved, care had to be taken to avoid patient rotation, since this too could alter the wave form.

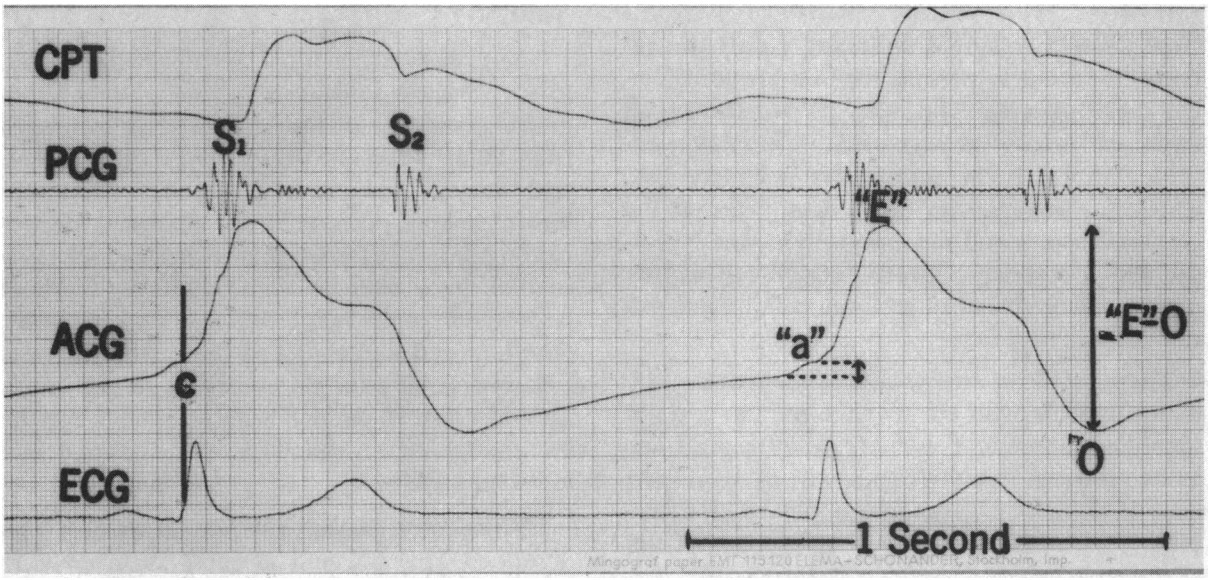

Figure 1.—The normal apexcardiogram (ACG). Note the acute downslope of the systolic wave beginning with the onset of ejection (E point) with a change in slope in the latter part of systole. The atrial filling wave ("a" wave) is quite small in amplitude in comparison to the vertical amplitude of the entire wave (E-O). Additional abbreviations: CPT= carotid pulse trace, PCG= phonocardiogram, ECG= electrocardiogram, O= opening of mitral valves, S_1= first heart sound, S_2= second heart sound, C= onset of ventricular contraction.

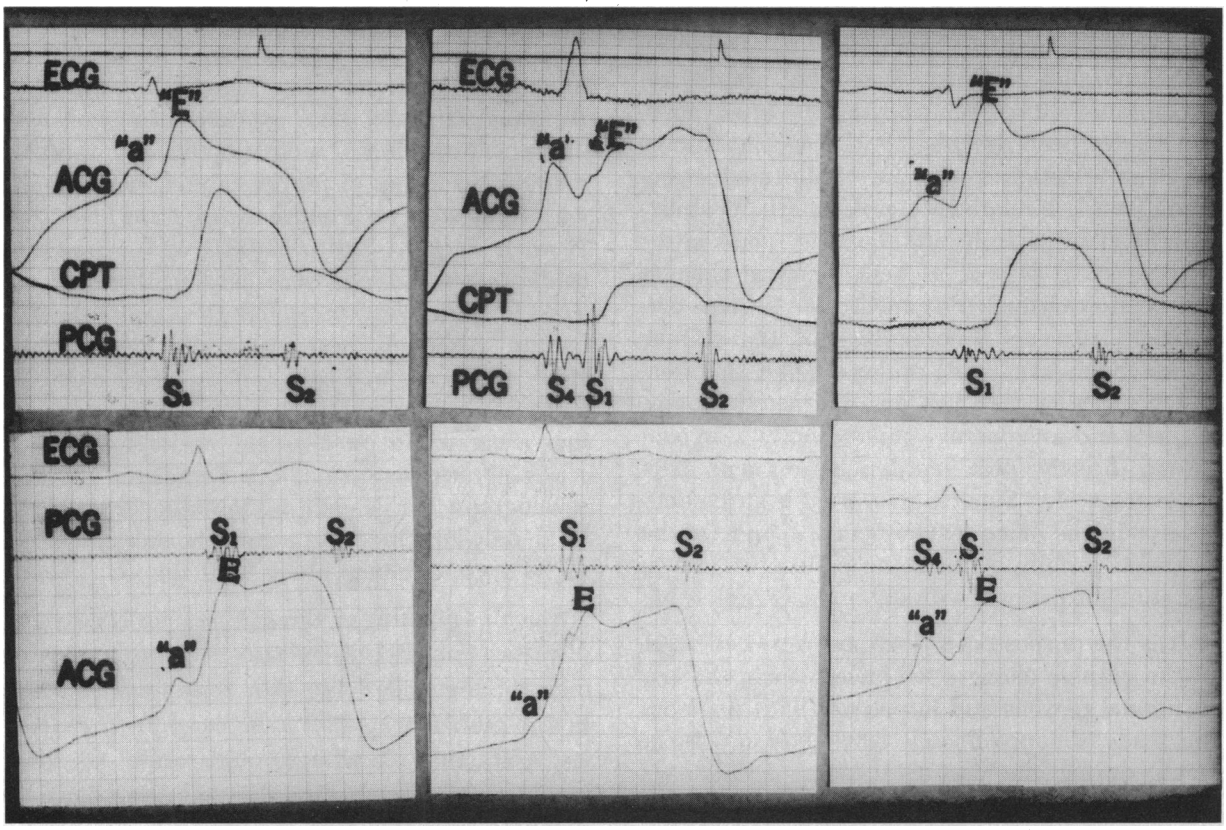

Figure 2.—ACG's recorded from patients with ischemic heart disease. In comparison to Figure 1 note the much larger "a" wave, a change in downslope angle of the systolic wave before midsystole to be followed by a more gradual downslope (top left), a paradoxical bulge (top, center and lower three strips) or a sustained wave (top right). Abbreviations: same as Figure 1.

Results

The normal ACG. Figure 1 shows an ACG recorded from a normal subject along with a simultaneous ECG, CPT and PCG. The initial upward deflection represents ventricular filling due to atrial contraction and is called the "a" wave. Its total amplitude was usually less than 10 percent of the entire systolic contraction wave (SCW) and its duration in 166 normal subjects was 64 ± 11 msec.

Beginning usually just before the peak of the R wave of the ECG, is a sharp upstroke (point C) signifying the onset of ventricular contraction. Note the onset of this upstroke begins on a horizontal plane above the onset of the ascending limb of the "a" wave. It is to be emphasized that the onset of the ventricular upstroke should not occur below the level of the onset of the "a" wave. The finding of such a "negative" wave means erroneous placement of the ACG pick-up, with resultant artifactual recordings.[46]

The ventricular upstroke reaches its own peak (E point) at the time of opening of the aortic valves shortly after mitral and tricuspid valve closure (S_1). At the onset of ejection, represented by the beginning upstroke of the carotid pulse tracing, there is an acute downstroke of the normal SCW of the ACG with a change in contour of the downslope beginning about mid-mechanical systole (interval between the first heart sound (S_1) and the second sound (S_2)).

Figure 2 shows a series of ACG's recorded from creased in both amplitude and duration and, patients with IHD. The "a" wave is frequently inwhen abnormal, is often a reflection of elevation of the left ventricular end diastolic pressure,[35-38] or increased left atrial filling pressure.

Distinct differences are noted as well in the SCW. The downslope is much more gradual; it may be sustained throughout systole or even may be paradoxical in direction. A change in contour of the downslope is often noted well before mid-mechanical systole. Such abnormal patterns of the downslope of the ACG probably represent external reflections of dyskinesis, akinesis, and asynchrony commonly noted in ventriculograms of patients with IHD. The SCW of the ACG, whether normal or abnormal, will remain stable when repetitive tracings are taken over a period of months to years, as long as technically proper

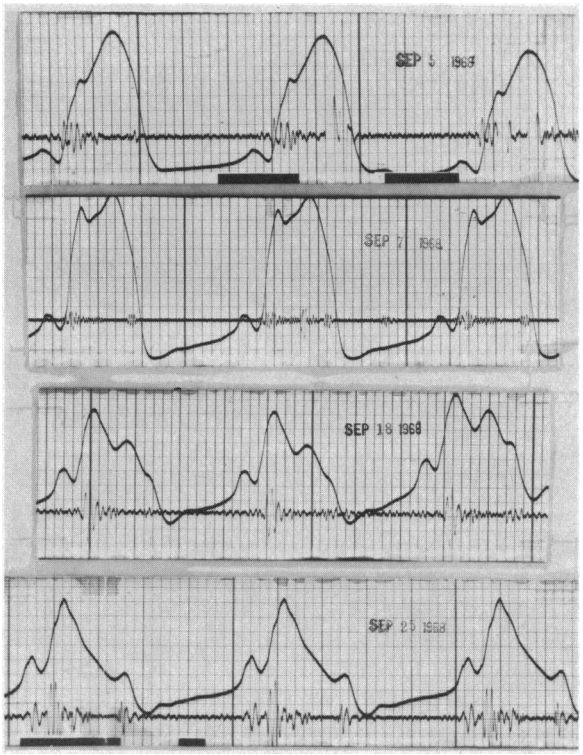

Figure 3.—Serial ACG's in a 62-year-old patient with an acute myocardial infarction whose initial ECG failed to show diagnostic changes. Note the pronounced paradoxical bulge in the initial recording with gradual recovery. Note too the increasing "a" wave amplitude with the development of a fourth heart sound.

recordings are obtained, and the patient's disease remains stable.[46]

The "a" wave duration in 215 patients with IHD was 73 ± 20 msec. Because of abnormal skewing of the data in IHD patients and in normal controls, using a standard "t" test, the differences between the means were significant ($P<.0005$).

Changes in Acute Myocardial Infarction. Figure 3 shows representative serial ACG's recorded in a 62-year-old man who had had myocardial infarction eight years previously and had remained asymptomatic for the next six years. On the morning of his admission to the hospital he had mild chest pain similar to that experienced during his first heart attack, and lasting about one hour. The symptoms were not accompanied by autonomic nervous system phenomena. The patient was seen three hours later and an electrocardiogram revealed only evidence of an old inferior wall infarction. The initial ACG is seen in the upper strip of Figure 3 and pronounced paradoxical bulging of the SCW is clearly evident.

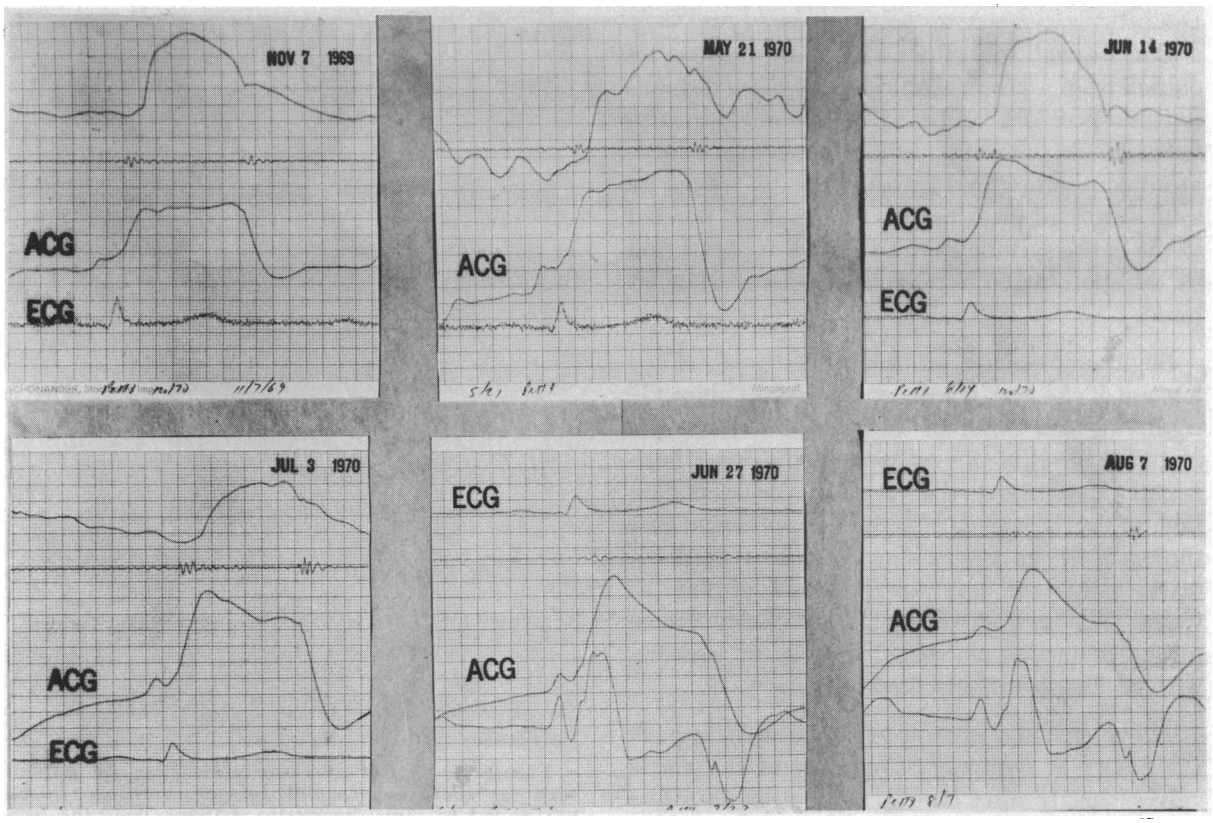

Figure 4.—Serial ACG's in 65-year-old patient with pre-infarction angina. Note the development of a paradoxical bulge and increased "a" wave amplitude in the recording of May 21 in comparison with November 7 when the patient was asymptomatic. Gradual recovery is seen in the follow-up recordings.

The patient reluctantly followed a recommendation of immediate admission to hospital. An hour later, while he was in the emergency room awaiting admission, cardiac arrest took place. He was successfully resuscitated. The serial changes in the SCW may be noted along with a large "a" wave presumably secondary to loss of ventricular compliance. The paradoxical bulge diminished gradually during the stay in hospital. The day following admission, evidence of a new inferior wall infarction was seen on the ECG and there was moderate enzyme elevation. Subsequent recovery was uneventful except for mild congestive heart failure. A fourth heart sound coinciding with the "a" wave is seen best in the bottom strip of Figure 3.

It is common knowledge that the initial ECG may fail to show changes during the early hours and even days of an acute infarction. It has been our experience that the ACG is almost always abnormal at the onset of an infarction and changes progressively during the first several days.[44,45] Interestingly, paradoxical bulges of the SCW as well as an "a" wave of increased amplitude were noted regardless of whether the infarction was inferior, anterior or subendocardial. It would seem that the abnormal wall motion occurring during an acute infarction, whatever its location, was transmitted in some manner to the apex impulse.

Transient abnormalities were often seen in the presence of simple myocardial ischemia when the ECG showed only T wave changes. This suggests the ACG may be more sensitive in detecting left ventricular dysfunction. Similar findings have been noted by other investigators, in that abnormalities of the precordial impulse frequently antedate other evidence of left ventricular disease.[47,48] The importance of this cannot be overemphasized since the ECG provides anatomical and not functional information. More complete details will be reported separately.[49]

Pre-infarction angina. Figure 4 shows serial tracings in a 65-year-old woman who had symptoms highly suggestive of pre-infarction angina. She described increasing fatigue, dyspnea on ex-

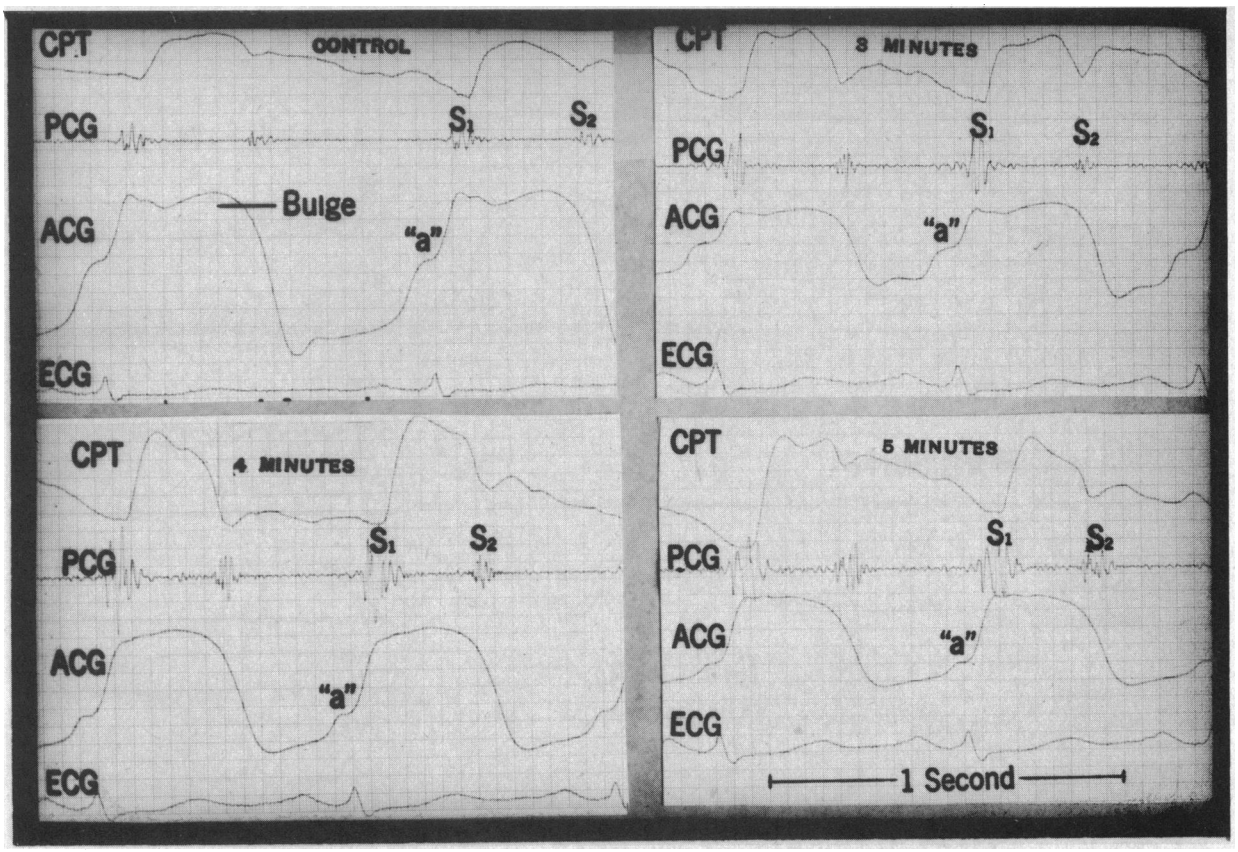

Figure 5.—Serial ACG's during angina pectoris with recovery following nitrogylcerine administration. Note the giant "a" wave and bulge of the systolic wave initially.

ertion, palpitation and angina pectoris. Of great significance was the fact these symptoms were not volunteered, indeed were actually denied in written answers to questions on the history form and only came to light on close re-questioning of the patient after the apexcardiographic changes of May 21. Serial ACG's in the months preceding May 21, although abnormal as shown in the recording of 7 November 1969, had remained stable. Note in the tracing of May 21, that the "a" wave amplitude, expressed as a relative percentage of the entire SCW, had gone from 13 percent on November 7 to 24 percent on May 21 while the SCW now showed a distinct paradoxical bulge that could not be eliminated despite repeated repositioning of the ACG pick-up and rotation of the patient. Subsequently, the patient admitted to the new development of the above symptoms but said she had attributed them to "old age." The progressive improvement of the ACG in the ensuing weeks is evident. It was characterized by a reduction in the amplitude of the "a" wave, loss of the paradoxical bulge, and improvement in the downslope of the SCW. Serial ECG's during this period all remained within normal limits. Coinciding with the improved ACG was a gradual disappearance of the previously noted symptoms. In a series of 20 such patients with characteristic pre-infarction angina, only eight (40 percent) showed changes in their ECG while all 20 showed progressive changes in their ACG similar to the change depicted in Figure 4. In contrast to acute infarction, the ACG changes in pre-infarction angina occurred over a more prolonged period.

Changes Noted in Angina Pectoris. Figure 5 shows serial ACG's obtained from a 60-year-old man during angina. The patient had documented occlusion of the right coronary artery, 90 percent occlusion of the left circumflex and a ventricular aneurysm. Notable in the initial strip was a giant "a" wave as well as significant paradoxical bulging of the SCW. Beginning about three minutes after the sublingual administration of nitroglycerine, reduction in the amplitude of both the bulge and "a" wave was seen. By five minutes, "a" wave amplitude had been reduced from 39 percent to 15 percent. The SCW, although still abnormal, showed loss of the bulge. These serial

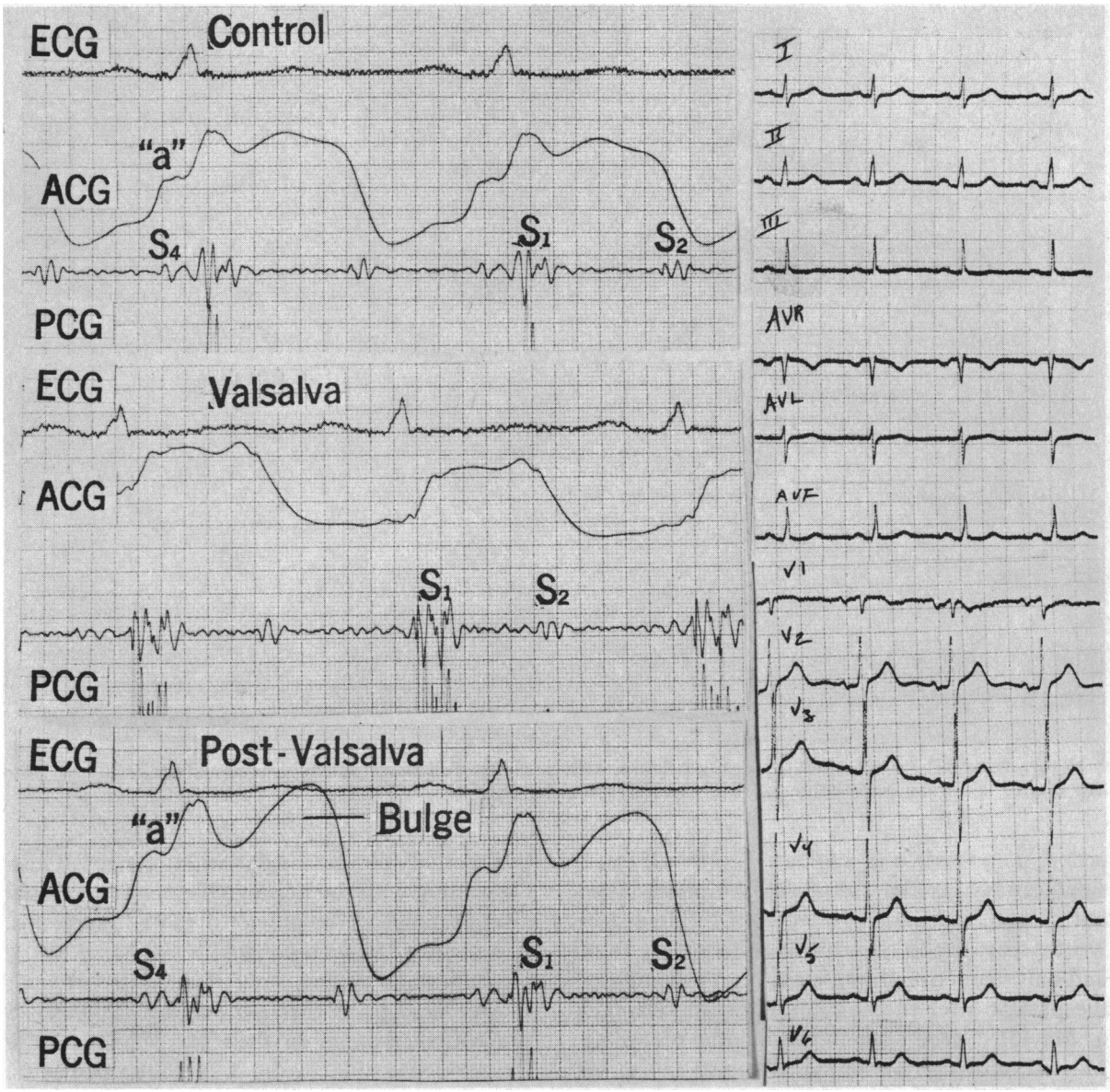

Figure 6.—ACG recorded in a 55-year-old man with unsuspected heart disease. Note the disappearance of the large "a" wave, bulge and S_4 during the Valsalva maneuver, with intensification immediately on recovery. The EKG on the right, taken a few moments later, was normal.

tracings were obtained without movement of the ACG pick-up or rotation of the patient.

Although the presence of disease was later confirmed in this patient by direct cardiac catheterization, it is reasonable to assume that serial changes in the ACG, similar to those described in Figure 5, in the absence of patient rotation and apex pick-up displacement, are acceptable documentation of changing left ventricular function compatible with the hemodynamic changes occurring in angina pectoris.

In our serial studies of patients with IHD, paradoxical bulges were often noted in patients during angina. Commonly, patients would show the presence of a bulge before the development of pain, but often no word of pain would be volunteered or even given in reply to specific questioning. The use of nitroglycerine during these episodes would cause prompt abatement of these abnormalities within the space of a few minutes. The resting ECG was normal in 12 of 30 patients who had frequent angina pectoris—several times a day or week. In contrast, the resting ACG was considered abnormal in all 30 patients.

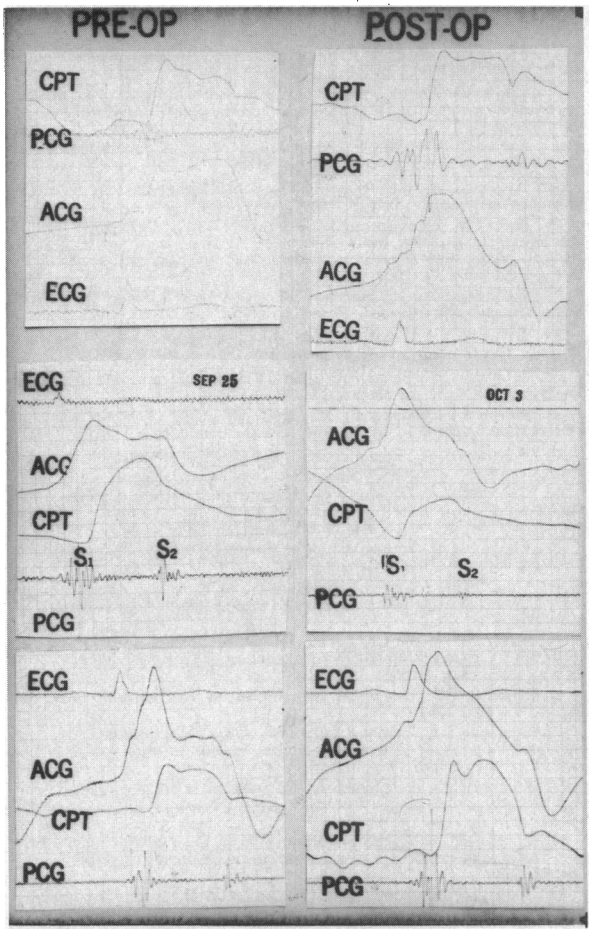

Figure 7.—ACG's before coronary by-pass operation (left hand strips) and after operation (right hand strips) in three patients. Note improved systolic wave in the postoperative tracings.

Another method of identifying abnormalities of left ventricular wall motion is seen in Figure 6. The patient, a 55-year-old man, was known to have mild diabetes, controlled with oral agents, but was otherwise reported to be in good health. A recent complete physical examination by an internist and an ECG had disclosed no abnormality. The initial recording in the top strip of Figure 6 shows a very large "a" wave, a paradoxical bulge of the SCW and a high frequency fourth heart sound (recorded up to 100 Hz). A Valsalva maneuver caused pronounced reduction in all three abnormalities. Upon Valsalva release, striking intensification of all abnormalities took place. An ECG taken immediately after release was entirely normal. Interestingly, no chest discomfort was experienced by the patient although apprehension was evident. Again, it is reasonable to assume that when such changes occur as part of a serial sequence, abnormal left ventricular function may be presumed to be present.

Pre-Coronary and Post-Coronary By-pass Changes. Figure 7 shows tracings of three patients with documented IHD, established by coronary cinearteriography, and considered surgical candidates for a by-pass graft. The strips show the pre-operative abnormalities and tracings taken shortly after corrective coronary by-pass operation was successfully carried out in each patient. The improvement in the SCW of the ACG consists of replacement of the paradoxical or sustained contraction wave by a rapid downslope throughout all of systole. In each instance the improved ACG was associated with loss of angina, easy fatigability and other symptoms present prior to surgery.

Discussion

Convincing data now seems to be present that the ACG is a sensitive indicator of the abnormal left ventricular wall function known to be present in most patients with ischemic heart disease. Since abnormal myocardium contracts at reduced velocity while maintaining an elevated wall tension,[50] the slower downslope of the abnormal ACG appears to reflect this reduction of velocity. Similarly, paradoxical bulging of the SCW apparently reflects paradoxical wall motion.[40] Initially, normal and abnormal myocardium both begin their shortening simultaneously; however, normal myocardium presumably completes its contraction phase first, apparently before mid-systole, while the slower, abnormal myocardium is still shortening. That is to say, abnormal myocardium contracts out of phase (asynchrony).[51] This perhaps explains the abrupt change in contour of the SCW of the ACG before mid-systole in the abnormal patient. It is possible, therefore, that the ACG reflects the moment to moment contactile pattern of the left ventricle. In other words, it appears to be an externally recorded, reciprocal analog curve of a ventriculogram. Frame by frame analysis of the volume relations of a ventriculogram related to time, shows close similarity between the derived curves and the SCW of the ACG, both in the normal and the diseased patient.[50] There is a clear distinction, however. While the ventriculogram will localize the exact anatomic portion of the left ventricle, that is dyskinetic, such localization is, of course, not

possible with the ACG. As previously indicated, abnormal SCW's were seen regardless of the location of the infarction and even when myocardial ischemia was present. Perhaps the paradoxical bulge seen in the SCW is merely the late transmission of the impulse generated by the abnormally contracting myocardium.

More important than the ability of the ACG to reflect left ventricular dysfunction is its apparent enhanced sensitivity over the resting ECG.[44-48] Such sensitivity will be of extreme usefulness in patients with progressive symptoms when one cannot perform an exercise ECG. Additionally, the fact that the changes noted in the ACG in patients with acute myocardial infarction, pre-infarction angina, and angina pectoris seem to coincide with the clinical events makes this testing procedure especially attractive, particularly since it can be performed serially and as often as necessary without discomfort to the patient.

It is hoped, therefore, that the ACG will be performed more routinely in patients suspected of having IHD or pre-infarction symptoms when routine tests are non-diagnostic. Whether recorded as a single tracing, and especially when recorded serially, it will provide functional and hemodynamic information that is not possible to obtain from an ordinary ECG. More frequent use of apexcardiography should allow earlier identification of IHD, permit earlier and more rational institution of prophylactic treatment and so help reduce the present high morbidity and mortality from this illness.

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